Polybrominated Diphenyl Ethers in House Dust and Clothes Dryer Lint

HEATHER M. STAPLETON,*,† NATHAN G. DODDER,† JOHN H. OFFENBERG,‡ MICHELE M. SCHANTZ,† AND STEPHEN A. WISE†

National Institute of Standards and Technology, 100 Bureau Drive, Mailstop 8392, Gaithersburg, Maryland 20899, and Environmental Protection Agency, Human Exposure and Atmospheric Sciences Division, Research Triangle Park, North Carolina 27711

Few studies have measured the flame retardants polybrominated diphenyl ethers (PBDEs) in the indoor environment. Here, we report measurements of PBDEs in house dust samples collected from the Washington, D.C. metropolitan area in the United States. Dust samples were analyzed for 22 individual PBDE congeners and our results found PBDEs present in every sample. Concentrations of total PBDEs ranged from 780 ng/g dry mass to 30 100 ng/g dry mass. The dominant congeners observed in the dust samples were congeners associated with the pentaBDE and decaBDE commercial mixtures. Ancillary data were collected on the homes and examined for any correlations with total PBDE concentrations. No correlations were observed with year of house construction, type of flooring (i.e., hardwood vs carpet) or the number of television sets or personal computers in the home. However, a significant inverse correlation ($p < 0.05$) was observed between the area of the home and the contribution of BDE 209 to the total PBDE concentration in dust. Using estimates of inadvertent dust ingestion (0.02–0.2 g/day) by young children (ages 1–4), we estimate ingestion of total PBDEs to range from 120 to 6000 ng/day. Clothes dryer lint was also sampled and analyzed for PBDEs from five of the homes and were present in all five samples ranging from 480 to 3080 ng/g dry mass. This study demonstrates that PBDEs are prevalent at relatively high concentrations within homes where people, and particularly young children, may be susceptible to exposure.

Introduction

Polybrominated diphenyl ethers (PBDEs) are brominated flame retardant chemicals that are applied to many common products found within homes such as furniture, carpeting, mattresses, televisions, coffee makers and hair dryers (*1*). Because of fire safety standards, many of these products are required to contain flame retardant chemicals which significantly delay the onset and spread of fire. The use of flame retardants in these products is estimated to have helped save millions of dollars in property damage and to have saved many lives (*2*). However, the heavy use of these chemicals, and the manner in which they are applied, has caused PBDEs to leach from the treated materials and accumulate in animals and humans. Reports have now demonstrated that concentrations of PBDEs have been increasing rapidly over the past 25 years in many environmental matrixes (*3*, *4*) and in human serum and breast milk (*5*–*7*).

PBDE concentrations have been measured in human serum, adipose tissue, and breast milk and all have shown that PBDE levels are about 17 times higher in North American individuals than those from individuals in Europe (*8*). The European Union has phased out the use of two of the three commercial mixtures of PBDEs (pentaBDE and octaBDE) this year and the state of California will follow in 2008. Because of this phase out, North America now uses 98% of the world market demand for pentaBDE, the commercial mixture which contains the congeners that are commonly observed in human tissues. Great Lakes Chemical Co., the sole producer of pentaBDE in the United States, has chosen to voluntarily phase out the production of pentaBDE by the end of 2004 (*9*); however, products that contain pentaBDE (i.e., furniture and carpets) will most likely remain in most homes for years. The third commercial mixture, decaBDE, is still used without regulation. It is the most heavily used with an estimated world market demand of 56 000 tons as reported in 2001 (*10*). DecaBDE is usually incorporated into high-impact polystyrene that is commonly found in the casings of TV sets and computers.

Studies have suggested that dietary exposure is the most likely route by which people accumulate PBDEs (*11*). PBDEs are similar in structure to polychlorinated biphenyls (PCBs), which accumulate through dietary ingestion (*12*). However, in contrast to PBDEs, PCBs were used primarily in electrical transformers and capacitors that were external to the home environment. Leaching of PCBs from transformers in landfills, industrial regions, and at point sources all contributed to high levels of PCBs in the environment and their biomagnification in aquatic food chains. PBDEs, in contrast, are applied to products found in almost every home in percentages as great as 30% by mass of the product to which they are applied (*1*). Studies comparing indoor versus outdoor air have shown that concentrations are greater in homes (1.6–43 times greater) than they are in the outdoor environment (*13*). The large concentration of PBDEs in residential use products suggests that the home environment may also be a significant source of human exposure to PBDEs.

In the present study, we collected dust from 16 homes in the Washington, D.C. metropolitan area and one home in Charleston, SC, to analyze for PBDEs. Dust has been used as an indictor of indoor exposure to pollutants such as lead and pesticides (*14*, *15*). Young children are particularly vulnerable to contaminants found in dust as they are often in close contact with floors and dusty surfaces and have a greater propensity to put their hands and objects in their mouths. Past studies have set a precedent for contaminant exposure via dust in young children, as house dust has been positively correlated with children's blood lead levels (*15*–*17*).

PBDE congeners found in the pentaBDE commercial mixture cause neurobehavioral deficits and disrupt thyroid hormone homeostasis in rodent and human in vitro studies (*18*–*21*). Fewer studies have been conducted examining the toxicity of decaBDE; however, some studies using mice and

* Corresponding author phone: (301)975-8578; fax: (301)977-0685; e-mail: heather.stapleton@nist.gov.
† National Institute of Standards and Technology.
‡ Environmental Protection Agency.

10.1021/es0486824 Not subject to U.S. Copyright. Publ. 2005 Am. Chem. Soc. Published on Web 12/29/2004

TABLE 1. Ancillary Information Collected from Each House or Apartment Sampled

sample	city	type[a]	area (m^2)	year of construction	floor type[b]	no. of foam pieces	no. of TVs	no. of computers	hours computer used/week
1	California, MD	H	213.7	1987	H	9	2	1	168
2	Gaithersburg, MD	H	204.4	1974	H	5	2	0	0
3	Lusby, MD	H	185.8	1992	C	3	2	1	15
4	Gaithersburg, MD	H	185.8	1967	H	2	4	3	50
5	Lusby, MD	H	167.2	1986	C	3	1	3	168
6	Lusby, MD	H	167.2	2002	C	3	2	1	2
7	Silver Spring, MD	H	157.9	1949	H	13	2	2	6
8	Charleston, SC	H	116.1	1994	C	2	1	1	168
9	Silver Spring, MD	T	223	1960	H	3	1	1	15
10	Arlington, VA	T	139.4	1983	H	3	2	1	7
11	Silver Spring, MD	B	65	1923	C	4	1	1	14
12	Lusby, MD	B	55.7	1987	C	2	1	0	0
13	Oakton, VA	A	101.3	1984	C	2	2	2	80
14	Alexandria, VA	A	83.6	1989	C	3	1	1	168
15	Germantown, MD	A	80.8	1987	C	2	1	1	10
16	Gaithersburg, MD	A	62.2	2000	C	1	1	1	7
17	Washington, D.C.	A	55.7	1960	C	1	1	1	4

[a] Key to type of home: H (single-family detached home), T (townhouse), B (basement apartment), A (one- or two-bedroom apartment). [b] Flooring type: C (carpeted), H (hardwood floors with area rugs).

rats suggest that chronic exposure may lead to neoplastic nodules in the liver and increases in the incidence of hepatocellular adenomas and carcinomas (*22*, *23*). Very little is known about the direct effects of chronic PBDE exposure on people and particularly on babies and young children who are in sensitive developmental stages. Our objectives in this study were to measure PBDEs in house dust and determine the potential for exposure of PBDEs to young children via house dust. In addition, clothes dryer lint was analyzed for PBDEs as an alternate matrix for assessing the levels of PBDEs within the home and to determine if PBDEs may be adsorbing to clothes.

Materials and Methods

Dust samples were collected from 16 homes in the Washington, D.C. metropolitan area and one home in Charleston, SC (Table 1), between January and March of 2004. Dust was collected from the floor in the main family room of all homes with a small commercial vacuum (Euro-Pro model, 900 W) equipped with a hose. Dust entering the vacuum first passed through a 1-mm wire mesh before being collected on a standard coffee filter (white-bleached filter for 12-cup drip coffee maker) which was inserted between the basket and HEPA filter. Between sample collections, the vacuum was thoroughly cleaned with hot water and a methanol rinse, and the coffee filter was replaced. In each home, dust was collected by vacuuming the rugs or hardwood floors (most with area rugs) until sufficient mass (0.1−0.5 g) was collected on the filter (approximately 15−30 min of vacuuming). The dust samples that were collected were assumed to be dry and no measurement of water content was performed. In place of a field blank, sodium sulfate powder was spread across a tile floor and collected with the vacuum in a manner similar to the dust samples. After vacuuming, the dust or field blank was scraped off the filter into precleaned glass jars using methanol-rinsed spatulas and taken back to the laboratory for extraction. Lint was collected from homes by removing the lint trap from the clothes dryer and collecting all the material off the lint trap, which was then wrapped in clean aluminum foil and placed in a plastic bag until extraction.

An occupant from each home was asked to complete a survey regarding some ancillary parameters of the home. Data collected from each home included year of construction, area, the number of foam-containing couches and chairs, the number of TVs and computers, and the number of hours each week the computer was left on. Observations were also made regarding whether the home contained predominantly hardwood or carpeted floors in the main family room.

Prior to extraction, between 50 and 100 ng (in hexane) of a ^{13}C-labeled BDE 209 (2,2′,3,3′,4,4′,5,5′,6,6′-decabromodiphenyl ether, BDE 209L) and a ^{13}C-labeled chlorinated diphenyl ether (2,2′,3,4,5-pentachlordiphenyl ether, CDE 86L, both from Cambridge Isotope Laboratories, Andover, MA) were added to each sample as internal standards. Dust samples were extracted using pressurized fluid extraction (Dionex model ASE 200) with dichloromethane. All samples, blanks, and calibration solutions were extracted using the following program parameters: temperature at 100 °C, heat time for 5 min, static time for 5 min, and pressure at 13.8 MPa (2000 psig), for three cycles. The extract was reduced in volume to 0.5 mL using an automated evaporation system and solvent exchanged to hexane. The extract was further cleaned using precleaned silica Sep-Pak cartridges (Waters Co., Milford, MA). Cartridges were first cleaned with 10 mL of hexane and the extract was eluted using 20 mL hexane. After concentration to 0.5 mL, the final extract was measured for PBDEs using an Agilent 6890 series gas chromatograph coupled to an Agilent 5973 mass spectrometer (GC/MS).

Quantification of BDE congeners was performed with a GC/MS using negative chemical ionization and was operated in the selected ion monitoring mode. All BDEs were quantified using ions 79 and 81 (bromide ions) with the exception of BDE 209, which was monitored with ions 487 and 409. The 22 individual BDE congeners that were quantified in this study include triBDEs: 17, 28; tetraBDEs: 71, 47, 66; pentaBDEs: 100, 99, 85; hexaBDEs: 154, 153, 138, 156; heptaBDEs: 184, 183, 191, 190; octaBDEs: 197, 196; nonaBDES: 208, 207, 206, and the fully brominated BDE 209. A 15 m × 0.25 mm (i.d.) 5% phenyl methylpolysiloxane capillary column (0.25-μm film thickness; J & W Scientific) was used for the separation of the BDE congeners, and all injections were performed with cool on-column injection. The inlet was programmed to follow the oven temperature program which was 80 °C for 2 min followed by a temperature ramp of 12 °C/min to 140 °C, then another ramp from 140 °C to 280 °C at 5 °C/min (held for an additional 20 min at 280 °C). The ion source was held at a constant temperature of 200 °C, the quadrupole was held at 100 °C, and the temperature of the transfer line was held at 280 °C. The method detection

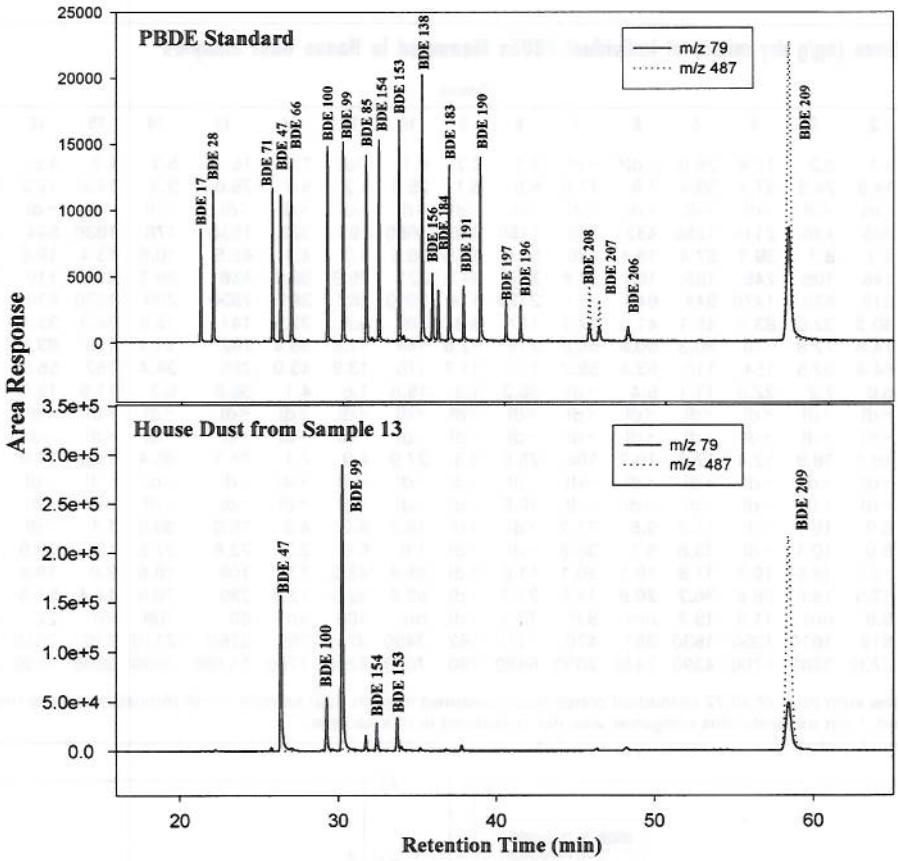

FIGURE 1. GC/ECNI-MS chromatogram comparison of PBDE standard and house dust collected from sample 13. Ions 79 and 81 were used to monitor tri- through nonaBDEs, while ion 487 and 409 were used to monitor BDE 209.

limit (MDL) for the PBDE congeners ranged from 1 ng/g dry mass (for BDE 28) to 6 ng/g dry mass (for BDE 209). Instrumental detection limits ($S/N = 50$) for BDE congeners ranged from 0.1 to 0.4 ng. Field blanks contained traces of BDEs 47, 99, and 209 but all values were low enough (<5 ng), relative to the smallest mass measured (78, 38, and 55 ng for BDEs 47, 99, and 209, respectively) to avoid blank subtraction. Replicate extractions were performed on three of the house dust samples and values for all replicates were within 25% of each other. The National Institute of Standards and Technology (NIST) has an indoor dust standard reference materials (SRM 2585) that is in the process of being certified for PBDEs and which will be available in 2005 (*24*). These SRM was used as quality control material on the values reported in this paper.

Results and Discussion

Concentrations in Dust. Each house sampled has been given a number and is listed in Table 1 with the ancillary information collected from the survey of each home. A representative house dust chromatogram displaying the peaks for PBDEs is presented in Figure 1. PBDEs were detected in every house dust sample collected and concentrations of total PBDEs (sum of all 22 individual congeners) ranged from 780 to 30 100 ng/g dry mass (Table 2). This range in concentration is very similar to a study recently conducted by the Environmental Working Group in which they sampled house dust from 10 homes around the United States and observed concentrations ranging from 600 to 41 000 ng/g dry mass (*25*). In another study, Rudel et al. (*26*) collected dust samples from 89 homes in the state of Massachusetts and measured only three BDE congeners (BDE 47, BDE 99, and BDE 100) and found average concentrations of 700, 1290, and 170 ng/g dry mass for BDE 47, BDE 99, and BDE 100, respectively. In a study conducted in Germany (*27*), PBDEs were measured in house dust from 40 German homes between 2001 and 2003 and the average concentrations were 120, 180, and 20 ng/g dry mass for BDE 47, BDE 99, and BDE 100, respectively. Comparing these results to this study, the concentrations of these three congeners are almost an order of magnitude higher in house dust found in U. S. homes relative to homes in the European Union, similar to the trend observed for PBDEs in human serum and breast milk, and in fish (*8*).

Concentrations of total PBDEs measured in the Washington, DC, area house dust samples are relatively high compared to most sediment samples collected in various countries. Total PBDEs in most sediment samples (both United States and abroad) are typically less than 100 ng/g dry mass (*8*) with the few exceptions measured at industrial point sources or near wastewater treatment plants (*28, 29*). The highest concentrations of PBDEs in abiotic samples are often found in sewage sludge. The total concentration of BDE 47, BDE 99, and BDE 100 was measured in sewage sludge from 11 areas around the United States and concentrations ranged from 1120 to 2290 ng/g dry mass (*30*), which were lower than the average concentration for these three congeners in the house dust measured. BDE 209 was also measured in these sewage sludge samples (*30*), and it ranged from 85 to 4890 ng/g dry mass, comparable to levels in house dust.

Commercial BDE Composition in Dust. As mentioned previously, three PBDE commercial mixtures are used on different types of products found within homes. The pentaBDE mixture is predominantly applied to polyurethane foam found within many couches and chairs. The dominant BDE congeners in pentaBDE mixtures are BDE 47, BDE 99, and BDE 100, which are the most abundant congeners in human

TABLE 2. Concentrations (ng/g dry mass) of Individual PBDEs Measured in House Dust Samples[a]

	house																		
congener	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	mean	median
BDE 17	21.6	4.1	6.3	17.4	25.0	<dl[a]	<dl	2.1	2.2	6.1	<dl	1.5	15. 0	5.2	4.2	4.5	<dl	8.9	4.2
BDE 33, 28	76.5	14.9	24.3	37.1	59.4	7.9	17.0	8.9	6.1	25.0	4.2	5.4	25.0	9.3	14.8	12.9	2.9	20.7	14.8
BDE 71	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl
BDE 47	7610	885	436	2110	1250	432	388	1150	356	1650	103	325	1830	178	1030	644	352	1220	644
BDE 66	142	9.3	8.7	39.1	57.4	18.4	<dl	59.6	12.9	30.5	5.3	4.9	45.5	10.6	13.4	19.6	7.5	28.5	13.4
BDE 100	2090	145	108	249	165	108	94.6	367	34.5	321	25.9	59.9	436	39.3	215	119	77.0	274	119
BDE 99	13,800	819	676	1470	944	648	571	2120	174	1910	162	381	2690	209	1330	610	444	1700	676
BDE 85	620	30.3	32.0	83.1	45.1	41.0	27.7	117	6.8	103	5.8	22.5	141	12.8	74.3	33.1	19.2	83.4	33.1
BDE 154	1250	74.8	72.8	116	80.3	50.8	65.0	214	12.6	144	11.8	35.4	282	31.1	110	63.8	34.7	156	72.8
BDE 153	1510	64.4	92.5	154	110	63.4	59.2	217	11.7	170	13.9	43.0	286	34.4	152	55.9	38.0	181	64.4
BDE 138	111	6.0	7.2	22.0	11.1	5.4	<dl	36.2	1.3	15.8	1.6	4.1	36.6	6.2	11.6	12.2	5.2	17.3	7.2
BDE 156	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl
BDE 184	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl
BDE 183	71.5	10.1	18.9	12.4	17.6	19.2	168	25.5	1.3	27.9	4.9	7.1	24.1	65.4	15.8	14.9	17.6	30.7	17.6
BDE 191	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl
BDE 190	3.0	<dl	1.0	<dl	<dl	<dl	<dl	10.5	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	<dl	4.5	<dl
BDE 197	22.4	6.9	15.7	<dl	12.2	9.5	77.2	<dl	<dl	14.7	5.0	4.8	15.0	39.8	7.1	<dl	13.5	17.4	9.5
BDE 196	16.2	5.0	10.1	<dl	13.8	6.7	38.6	<dl	<dl	7.6	6.0	2.7	22.6	27.5	4.5	20.5	21.4	14.5	7.6
BDE 207	34.1	10.1	18.5	10.1	17.8	19.1	30.1	11.0	<dl	35.4	42.0	7.9	109	76.6	9.8	19.8	29.1	30.0	19.1
BDE 206	58.9	13.9	18.0	28.6	36.2	20.8	13.5	21.7	<dl	52.2	92.5	12.0	239	76.9	14.4	51.6	68.2	51.1	28.6
BDE 208	nm[b]	9.8	nm	11.8	19.2	nm	9.0	13.3	<dl	nm	108	nm	60	106	nm	22	22	34.7	19.2
BDE 209	2400	616	1010	1350	1530	951	470	1170	162	2490	3740	769	8750	2120	770	2910	4310	2090	1350
total	30,100	2730	2580	5780	4390	2440	2030	5480	780	7080	4250	1700	14,990	3030	3800	4590	5470	5900	4250

[a] Total represents the sum total of all 22 individual congeners measured in each dust sample. [b] <dl indicates sample measurement was less than the detection limit. [c] nm indicates this congener was not measured in this sample.

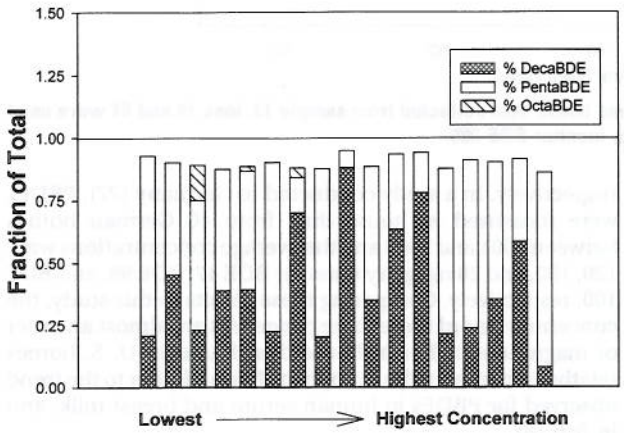

FIGURE 2. Contribution of commercial mixtures to the total BDE concentration measured in house dust samples. [% DecaBDE = % BDE 209/total BDE; % pentaBDE = (Σ BDE 47 + BDE 99 + BDE 100)/total BDE; % octaBDE = % BDE 183/total BDE].

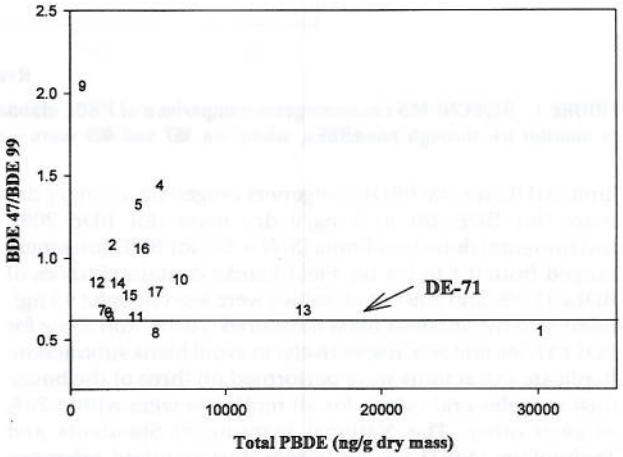

FIGURE 3. Ratio of BDE 47 to BDE 99 measured in all house dust samples versus the total BDE concentration. Line represents the ratio observed in the commercial pentaBDE mixture, DE-71. Numbers within the graph represent the sample number listed in Table 1.

tissues (*8*). The sum of these three congeners ranged from 290 to 23 500 ng/g dry mass in the house dust samples collected in the present study. As a percentage of the total PBDE burden, these pentaBDE congeners represented anywhere from 7 to 80% of the total (Figure 2); the average contribution to the total PBDE concentration was 48%.

BDE 209 is the dominant congener (97%) in the decaBDE commercial mixture. Concentrations of BDE 209 in house dust ranged from 162 to 8750 ng/g dry mass. As a percentage of the total PBDE burden, BDE 209 represented between 8 and 88% of the total (Figure 2), and the average contribution from decaBDE in these house dust samples was 41%. The variation in the contribution of BDE 209 to the total PBDEs among homes is surprising and may reflect different electronic products found within the homes (i.e., different brands of computers, stereo equipment, or other electrical appliances).

PentaBDE and decaBDE were the dominant mixtures measured in house dust samples in this study, although the third mixture, octaBDE, was detected at low levels in a few homes. OctaBDE consists primarily of BDE 183, BDE 197, and BDE 196. The contribution of these three congeners to the total PBDE burden in house dust samples ranged from <1% up to 14% (Figure 2).

BDE Ratios in House Dust. In the present study, we examined the ratio of BDE 47/BDE 99 in the house dust samples and compared it to the ratio in the commercial pentaBDE mixture. As seen in Figure 3, the ratio of BDE 47/BDE 99 in the commercial pentaBDE mixture, DE-71, is approximately 0.6. The two house dust samples that had the highest PBDE concentration displayed a ratio very similar to the commercial mixture. The house dust sample with the lowest PBDE concentration (780 ng/g dry mass) displayed a ratio that was the most aberrant from the commercial mixture ratio. Because the two house dust samples with the highest total PBDE concentration displayed a ratio similar to the technical pentaBDE mixture, it suggests that these dust samples were collected very close to a product that contained the pentaBDE mixture. The other house dust samples displayed a range (0.5−2.0) in the ratio of BDE 47/

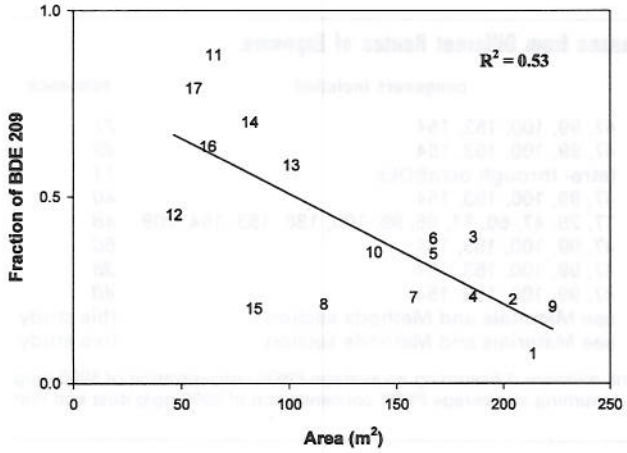

FIGURE 4. Correlation between area of the home sampled versus the contribution of BDE 209 to the total PBDE concentration measured in the house dust sample. ($p < 0.01$). Numbers within the graph represent the sample numbers listed in Table 1.

BDE 99, suggesting that other mechanisms were affecting the transport or accumulation of these congeners in house dust.

A few studies have demonstrated that BDE congeners debrominate either abiotically from exposure to UV light (*31*, *32*) or from apparent metabolic functions in fish (*33*). In the study by Eriksson et al. (*32*), photolytic breakdown of BDE 99 in methanol/water was observed with a half-life of approximately 64 days. However, the authors did not identify what products were formed from the decomposition of BDE 99. The removal of one of the metasubstituted bromine atoms from BDE 99 results in the formation of BDE 47. It is possible that degradation of BDE 99 could occur in the house dust samples, which would lead to the formation of BDE 47. This would result in the elevated ratios we observed in Figure 3; however, no studies have examined this issue. Alternatively, it is possible that BDE 47 may be fluxing out of the treated products at a greater rate than BDE 99, which would affect our observed ratios. BDE 47 has a higher vapor pressure than BDE 99 and modeling scenarios suggest that BDE 47 has a greater potential for long-range transport (and characteristic travel distance) relative to BDE 99 (*34*).

Factors Contributing to PBDEs in Homes. The owner of each home filled out a short questionnaire following sampling. Information such as year of house construction, area of the home, and number of TVs and computers present in the home were all collated and are presented in Table 1. Linear regression analysis indicated no significant associations between PBDE concentrations and the variables listed in Table 1, with one exception. There was a significant inverse relationship ($p < 0.05$) between the area of each home and the contribution of BDE 209 to the total PBDE concentration (Figure 4). Since data are only available for 17 house dust samples, it is difficult to determine if this correlation would hold true for all homes in the United States. Five apartments (area < 101 m^2) were sampled in this study and in four of these five apartments the contribution of BDE 209 to the total PBDE burden in house dust was 60% or greater. Two basement apartments were sampled and the contribution of BDE 209 in these two samples was 45% and 88%. In contrast, the contribution of BDE 209 in the townhomes and single-family detached homes ($n = 10$, area > 110 m^2) averaged 27 ± 10% of the total PBDE concentration. The explanation for this trend is unclear at this time.

PBDEs in Clothes Dryer Lint. To further examine the presence and concentrations of PBDEs in the home, 5 of the 17 homes sampled in this study were randomly revisited and lint samples were collected from the lint traps of the clothes dryer in the home. Presumably, the washing process may remove PBDEs prior to drying and it is not known how the drying heat affects the retention of PBDEs to clothes and lint.

TABLE 3. Concentrations of BDE Congeners (ng/g dry mass) Measured in Dryer Lint from Homes[a]

sample	BDE 47	BDE 100	BDE 99	BDE 183	BDE 209	total BDE in lint
10	145	23	105	7	104	480
15	130	24	133	3	230	570
16	210	54	280	1	58	680
13	310	58	370	2	800	1680
14	38	8	57	1	2890	3080

[a] Total BDEs represent the sum of 22 individual congeners measured in each sample. Sample number refers to the number assigned to each home sampled for dust.

Dryer lint has been examined in the past for lead levels and has been suggested as an alternative matrix for non-intrusive environmental contaminant screenings within the home (*35*). Dryer lint is a heterogeneous mixture of cotton and synthetic fibers, hair, and other materials and may be a sink for hydrophobic contaminants.

PBDEs were present in all five dryer lint samples in levels that were, on average, lower relative to the dust samples collected within the same home (480 ng/g to 3080 ng/g dry mass, Table 3). Four of the dryer lint samples were comprised of congeners from both the pentaBDE and decaBDE mixtures, while one lint sample from home 12 was 94% BDE 209. The source of the BDE 209 in this dryer lint sample is unknown but may come from contact with house dust. This same home had equivalent levels of PBDEs in both the house dust and dryer lint sample, and both dust and dryer lint had high contributions of BDE 209. There was no correlation between total PBDEs in dryer lint and total PBDEs in house dust in these five homes. However, there was a slight correlation ($p < 0.20$) in BDE 47 concentrations between dryer lint and house dust. Given that only five samples were used in this preliminary study, it is difficult to determine if the concentrations of pentaBDEs are correlated between house dust and clothes dryer lint. PBDEs in dryer lint may be more reflective of recent dryings and may depend on the material being dried (i.e., towels, sheets, or clothes), while house dust may be reflective of accumulation over longer periods of time. The source of the PBDEs in the dryer lint is unclear at this time but may suggest that our clothes are picking up PBDEs within the home which can then be collected in the dryer lint during the drying process. In contrast, we cannot rule out the possibility that PBDEs are present in the dryer itself. To the authors' knowledge, this is the first report in which PBDEs have been measured in dryer lint.

Potential Exposure via House Dust. Investigations into possible exposure routes have primarily focused on dietary intake, although scientists have stated that inhalation and dermal exposure in the home need to be further investigated (*36*); hence, the primary routes of PBDE exposure to people are still unclear. Some studies suggest a positive correlation between human BDE concentrations and fish consumption (*37*). However, other studies have found no such correlation between PBDEs in human milk versus dietary intake of fish (*38*).

Studies have demonstrated that concentrations of PBDEs are significantly higher in indoor air versus outdoor air (*13*) and indoor organic films on windows are also more concentrated in PBDEs as opposed to outdoor organic films (*39*). These studies suggest that indoor environments may be a significant pathway for exposure to PBDEs. One study in the United Kingdom estimated the relative intake of PBDEs from

TABLE 4. Comparison of PBDE Intake Rates among Different Age Classes from Different Routes of Exposure

exposure route	population	country	intake (ng/day)	congeners included	reference
diet	adults	Sweden	51	47, 99, 100, 153, 154	*21*
diet	adults	Finland	44	47, 99, 100, 153, 154	*49*
diet	adults	Spain	80–97	tetra- through octaBDEs	*11*
diet	adults	United Kingdom	107	47, 99, 100, 153, 154	*40*
nursing	newborns	United States	1770[a]	17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 209	*48*
nursing	newborns	United Kingdom	210[a]	47, 99, 100, 153, 154	*50*
nursing	newborns	Sweden	96[a]	47, 99, 100, 153, 154	*38*
inhalation	adults	United Kingdom	20	47, 99, 100, 153, 154	*40*
dust ingestion	children 1–4	United States	120–1180[b]	see Materials and Methods section	this study
dust ingestion	adults	United States	3.3[c]	see Materials and Methods section	this study

[a] Assuming milk sampled is 3% lipid and that a 5 kg infant ingests 800 mL milk/day. [b] Assuming an average PBDE concentration of 5900 ng/g dust and that children ingest between 0.02 and 0.2 g of dust/day (*14, 45*). [c] Assuming an average PBDE concentration of 5900 ng/g dust and that adults ingest 0.00056 g/day of dust (*51*).

diet versus inhalation on the basis of the concentrations of PBDEs measured in a few homes ($n = 7$) in England (*40*). Their study suggested that exposure via diet and inhalation was approximately 107 and 20 ng/day, respectively. However, no studies have examined the intake of PBDEs from house dust.

A few reviews have focused on house dust as a metric for assessing indoor exposure from organic contaminants, particularly in small children (*14, 41–43*). Studies have demonstrated that high lead levels in childrens' blood are positively correlated with lead levels in house dust, setting a precedent for house dust contamination and exposure to children (*15–17*). In a recent study, the "mouthing" of fingers by four-month-old infants was found to be responsible for as much as 2/3's of the infant's daily exposure to lead (*44*). Considering this, it may be necessary to assess the exposure of small children to PBDEs via house dust, particularly because the main source of these compounds is found in products within the home and is not tracked in from external environments. However, adequately assessing the exposure via house dust is confounded by many factors, including children's behavior and factors that affect the accumulation of PBDEs in house dust (i.e., frequency of vacuuming, types of products in the home, ventilation rates, etc.).

There are some simple models which have been used to estimate the daily intake of contaminants via house dust in young children (*14, 45*). These estimates assume that young children ingest anywhere from 0.02 to 0.2 g of dust daily. Using these estimates, we have provided a rough estimate of the daily intake of PBDEs in young children and adults via house dust on the basis of the average concentration of total PBDEs measured in house dust in this study (Table 4). Our exposure estimate for young children is approximately 120–1200 ng PBDEs daily, on the basis of the mean value of 5900 ng/g dust in these 17 house dust samples. However, if one uses the maximum concentration of PBDEs measured in this study (30 100 ng/g dry mass), intake of dust could be as much as 6000 ng/day. Considering one home among the 17 sampled displayed such a high concentration, it is possible that 5% of the general population might also display similar high concentrations of PBDEs in house dust.

Approximately 40% of this exposure estimate is attributable to ingestion of BDE 209 alone, the primary component of the decaBDE commercial mixture. Thus, dust exposure to small children may be a leading exposure route for decaBDE. BDE 209 is not typically measured (or is difficult to measure) in human tissues (breast milk, serum, and adipose tissue) and one study has shown that BDE 209 typically has a small half-life in human serum when it is present (*46*). As mentioned previously, some house dust samples were almost 90% BDE 209 and if one uses the maximum concentration of BDE 209 measured in house dust (8750 ng/g dry mass), intake of BDE 209 alone ranges from 180 to 1750 ng/day (assuming dust intake of 0.02–0.2 g/day). Our estimated intake of BDEs that are associated with the pentaBDE commercial mixture (i.e., BDE 47, BDE 99, and BDE 100) ranges from approximately 70–700 ng/day using the mean concentrations measured in house dust.

Table 4 compares the intake of PBDEs among different age groups from different exposure routes. It appears that young children in the United States may be exposed to higher levels of PBDEs through dust contact compared to European adults from either inhalation or dietary exposure. However, a few caveats must be taken into consideration with this comparison between the United States and European intake rates. The U.S. market demand for pentaBDE is approximately 95% of the worldwide market demand (*10*), which presumably leads to higher environmental levels of pentaBDE in the United States. Fish from the United States, for example, have BDE levels that are on average 9 times higher than in Europe (*8*). Therefore, humans in the United States may have a greater dietary intake of PBDEs than Europeans. It is unclear how the exposure of U.S. children to PBDEs from house dust compares to the dietary intake of PBDEs in adults from the United States. More studies are needed to estimate PBDE dietary intake in the U.S. population.

Studies have shown that babies accumulate PBDEs by both placental transfer from their mothers and from nursing on breast milk (*47, 48*). Considering this, it appears that infants and young children are receiving greater exposure from PBDEs through placental transfer, nursing on breast milk, and from inadvertent ingestion of house dust than adults do through their diet. This study highlights the need for more investigations into indoor exposure from PBDEs, particularly for young children. Concentrations of PBDEs in house dust were the same as levels measured in sewage sludge samples and are higher than concentrations measured in most sediments and soil. PBDEs were also present in dryer lint, which raises questions about the adsorption of PBDEs from products in our homes to our clothes.

Acknowledgments

H.M.S. and N.G.D. acknowledge financial support from the National Institute of Standards and Technology's National Research Council Postdoctoral Research program. The authors extend appreciation to Dr. Joel E. Baker and Bernie Crimmins at the Chesapeake Biological Laboratory for their discussions which led to this study. Certain commercial equipment, instruments, or materials are identified in this paper to specify adequately the experimental procedure. Such identification does not imply recommendation or endorsement by the National Institute of Standards and Technology, nor does it imply that the materials or equipment identified are necessarily the best available for the purpose. The United States Environmental Protection Agency through its Office

of Research and Development collaborated in the research described here. It has been subjected to Agency review and approved for publication.

Literature Cited

(1) *Environmental Health Criteria 162: Brominataed Diphenyl Ethers*; World Health Organization: Geneva, Switzerland, 1994.
(2) Bromine Science and Environmental Forum. http://www.bsef.com.
(3) Ikonomou, M. G.; Rayne, S.; Addison, R. F. *Environ. Sci. Technol.* **2002**, *36*, 1886−1892.
(4) Norstrom, R. J.; Simon, M.; Moisey, J.; Wakeford, B.; Weseloh, D. V. *Environ. Sci. Technol.* **2002**, *36*, 4783−4789.
(5) Norén, K.; Meironyté, D. *Chemosphere* **1998**, *40*, 1111−1123.
(6) Betts, K. *Environ. Sci. Technol.* **2002**, *36*, 50A−52A.
(7) Sjödin, A.; Jones, R. S.; Focant, J.; Lapeza, C.; Wang, R. Y.; McGahee E. E.; Zhang, Y.; Turner, W. E.; Slazyk, B.; Needham, L. L.; Patterson, D. G. *Environ. Health Perspect.* **2004**, *112*, 654−658.
(8) Hites, R. A. *Environ. Sci. Technol.* **2004**, *38*, 945−956.
(9) Tullo, A. *Chem. Eng. News* **2003**, Nov 10, 13.
(10) Major Brominated Flame Retardants Volume Estimates, BSEF 2003. http://www.bsef-site.com/docs/bfr_vols_2001.doc.
(11) Bocio, A.; Llobet, J. M.; Domingo, J. L.; Corbella, J.; Teixido, A.; Casa, C. *J. Agric. Food Chem.* **2003**, *51*, 3191−3195.
(12) Jensen, E.; Egan, S. K.; Canaday, R. A.; Bolger, P. M. *Toxicol. Ind. Health* **2001**, *17*, 157−162.
(13) Shoeib, M.; Harner, T.; Ikonomou, M.; Kannan, K. *Environ. Sci. Technol.* **2004**, *38*, 1313−1320.
(14) Roberts, J. W.; Dickey, P. *Rev. Environ. Contam. Toxicol.* **1995**, *143*, 59−78.
(15) Charney, E.; Sayre, J.; Coulter, M. *Pediatrics* **1980**, *65*, 226−231.
(16) Rhoads, G. C.; Ettinger, A. S.; Weisel, C. P.; Buckley, T. J.; Goldman, K. D.; Adgate, J.; Lioy, P. J. *Pediatrics* **1999**, *103*, 551−555.
(17) Sanchez-Nazario, E. E.; Mansilla-Rivera, I.; Derieux-Cortes, J. C.qq; Perez, C. M.; Rodriguez-Sierra, C. J. E. E. *P.R. Health Sci. J.* **2003**, *22*, 153−159.
(18) Fowles, J. R.; Fairbrother, A.; Baecher-Steppan, L.; Kerkvliet, N. I. *Toxicology* **1994**, *86*, 49−61.
(19) Eriksson, P.; Jakobsson, E.; Fredriksson, A. *Organohalogen Compd.* **1998**, *35*, 375−377.
(20) Meerts, I. A. T. M.; van Zanden, J. J.; Luijks, E. A. C.; van Leeuwen-Bol, I.; Marsh, G.; Jakobsson, E.; Bergman, Å.; Brouwer, A. *Toxicol. Sci.* **2000**, *56*, 95−104.
(21) Darnerud, P. O.; Gunnar, E. S.; Jóhannesson, T.; Larsen, P. B.; Viluksela, M. *Environ. Health Perspect.* **2001**, *109* (Suppl. 1), 49−68.
(22) Darnerud, P. O. *Environ. Int.* **2003**, *29*, 841−853.
(23) Birnbaum, L. S.; Staskal, D. F. *Environ. Health Perspect.* **2004**, *112* (1), 9−17.
(24) Stapleton, H. M.; Schantz, M. M.; Wise, S. A. In draft.
(25) Sharp, R.; Lunder, S. Report. 2004; http://www.ewg.org/reports/inthedust/index.php.
(26) Rudel, R. A.; Camann, D. E.; Spengler, J. D.; Korn, L. R.; Brody, J. G. *Environ. Sci. Technol.* **2003**, *37*, 4543−4553.
(27) Knoth, W.; Mann, W.; Meyer, R.; Nebhuth, J. *Organohalogen Cmpd.* **2003**, *2*, Section 1.
(28) Sellstrom, U.; Kierkegaard, A.; De Wit, C.; Jansson, B. *Environ. Toxicol. Chem.* **1998**, *17*, 1065−1072.
(29) Klosterhaus, S.; Stapleton, H. M.; Baker, J. E. Presented at the 3rd International Workshop on Brominated Flame Retardants, Toronto, Canada, 2004.
(30) Hale, R. C.; La Guardia, M. J.; Harvey, E. P.; Gaylor, M. O.; Matteson Mainor, T.; Duff, W. H. *Nature* **2001**, *412*, 140−141.
(31) Söderstrom, G.; Sellström, U.; De Wit, C.; Tysklind, M. *Environ. Sci. Technol.* **2004**, *38*, 127−132.
(32) Eriksson, J.; Green, N.; Marsh, G.; Bergman, Å. *Environ. Sci. Technol.* **2004**, *38*, 3119−3125.
(33) Stapleton, H. M.; Alaee, M.; Letcher, R. J.; Baker, J. E. *Environ. Sci. Technol.* **2004**, *38*, 112−119.
(34) Gouin, T. Harner, T. *Environ. Int.* **2003**, *29*, 717−724.
(35) Mahaffy, P. G.; Martin, N. I.; Newman, K. E.; Hohn, B.; Mikula, R. J.; Munoz, V. A. *Environ. Sci. Technol.* **1998**, *32*, 2467−2473.
(36) Sjödin, A.; Patterson, D. G.; Bergman, Å. *Environ. Int.* **2003**, *29*, 829−839.
(37) Ohta, S.; Ishizuka, D.; Nishimura, H.; Nakao, T.; Aozasa, O.; Shimidzu, Y.; Ochiai, F.; Kida, T.; Nishi, M.; Miyata, H. *Chemosphere* **2002**, *46*, 689−696.
(38) Lind, Y.; Darnerud, P. O.; Atuma, S.; Aune, M.; Becker, W.; Bjerselius, R.; Cnattingius, S.; Glynn, A. *Environ. Res.* **2003**, *93*, 186−194.
(39) Butt, C. M.; Diamond, M. L.; Truong, J.; Ikonomou, M. G.; ter Shure, A. F. H. *Environ. Sci. Technol.* **2004**, *38*, 724−731.
(40) Harrad, S.; Wijesekera, R.; Hunter, S.; Halliwell, C.; Baker, R. *Environ. Sci. Technol.* **2004**, *38*, 2345−2350.
(41) Lioy, P. J.; Freeman, N. C. G.; Millette, J. R. *Environ. Health Perspect.* **2002**, *110*, 969−983.
(42) Cohen Hubal, E. A.; Sheldon, L. S.; Burke, J. M.; McCurdy, T. R.; Berry, M. R.; Rigas, M. L.; Zartarian, V. G.; Freeman, N. C. G.*Environ. Health Perspect.* **2000**, *108*, 475−486.
(43) Butte, W.; Heinzow, B. *Rev. Environ. Contam. Toxicol.* **2002**, *175*, 1−46.
(44) Kranz, B. D.; Simon, D. L.; Leonardi, B. G. *J. Exposure Anal. Environ. Epidemiol.* **2004**, *14*, 300−311.
(45) United States Environmental Protection Agency. *Child-Specific Exposure Factors Handbook*; Interim Report, September 2002; National Center for Environmental Assessment: Washington, DC, 2002.
(46) Sjödin, A.; Hagmar, L.; Klasson-Wehler, E.; Dronholm-Diab, K.; Jakobsson, E.; Bergman, Å. *Environ. Health Perspect.* **1999**, *107*, 643−648.
(47) Mazdai, A.; Dodder, N. G.; Abernathy, M. P.; Hites, R. A.; Bigsby, R. M. *Environ. Health Perspect.* **2003**, *111*, 1249−1252.
(48) Schecter, A.; Pavuk, M.; Päpke, O.; Ryan, J. J.; Birnbaum, L.; Rosen, R. *Environ. Health Perspect.* **2003**, *111*, 1723−1729.
(49) Kiviranta, H.; Ovaskainen, M.; Vartiainen, T. *Environ. Int.* **2004**, *30*, 923−932.
(50) Kalantzi, O. I.; Martin, F. L.; Thomas, G. O.; Alcock, R. E.; Tang, H. R.; Drury, S. C.; Carmichael, P. L.; Nicholson, J. K.; Jones, K. C. *Environ. Health Perspect.* **2004**, *112*, 1085−1091.
(51) United States Environmental Protection Agency. *Exposure Factors Handbook* (*EFH*); EPA/600/C-99/001; National Center for Environmental Assessment, Office of Research and Assessment: Washington, DC, 1997.

Received for review August 22, 2004. Revised manuscript received October 19, 2004. Accepted November 10, 2004.

ES0486824